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RESEARCH ARTICLE

Is the Intraoperative Lactate–standard Base Excess Relationship Strong in Cardiac Surgery?

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ABSTRACT

Objectives: In this study, changes in lactate and standard base excess (SBE) values, which are indirect indicators of tissue perfusion, in patients undergoing cardiac surgery with cardiopulmonary bypass (CPB) were examined, and whether there was a correlation between them was investigated.

Methods: In total, 2,068 patients >18 years of age who underwent elective cardiac surgery with CPB were included in the study. Hemodynamic and blood gas parameters were recorded at four different time points: before and after anesthesia induction and at the beginning and end-of-extracorporeal circulation.

Results: Before anesthesia induction, the average lactate level was 1.0±0.5 mmol/L, and the average SBE level was 0.9±2.1. SBE and lactate had very weak correlations in all four intraoperative periods (r^2=0.01, r^2=0.02, r^2=0.06, r^2=0.06; p<0.001). At the end of the extracorporeal period, SBE and lactate were correlated (r^2=0.11; p<0.001) in patients with lactate values >2 mmol/L, but no correlation was found in patients with a lactate value <2 mmol/L (r^2=0.0003; p=0.526). The Kappa test revealed that lactate and SBE changes were incompatible in 274 (13.3%) patients. Hyperchloremia was detected in 1683 (94.5%) of 1781 patients in whom lactate increase and simultaneous SBE decrease were observed.

Conclusion: Lactate levels were weakly correlated with SBE throughout the intraoperative period. Because SBE is a calculated parameter and not a measured parameter, it should be evaluated together with lactate rather than interpreted alone.

Keywords: Cardiopulmonary bypass, lactate, standard base excess, tissue perfusion

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Introduction

In clinical practice, several hemodynamic and blood gas parameters are used to assess the adequacy of tissue perfusion.[1,2] Hemodynamic parameters and biomarkers, such as mean arterial blood pressure, cardiac output, heart rate, standard base excess (SBE), lactate, mixed or central venous oxygen saturation, and oxygen extraction ratio, which are considered indirect tissue perfusion indicators, are frequently used to evaluate tissue perfusion and microcirculation.[3]

These hemodynamic and blood gas parameters in clinical practice have limitations depending on changing conditions.[4,5] Because of these limitations, in clinical practice, multiple parameters are evaluated together to obtain information about tissue perfusion, especially during cardiac surgery with extracorporeal circulation (ECC) and in patients with high potential for microcirculation and macrocirculation disruption, such as those with sepsis and trauma.[3]

Hyperlactatemia due to microcirculatory and macrocirculatory hemodynamic disturbances after cardiac surgery with cardiopulmonary bypass (CPB) is known to be associated with poor outcomes, but it is not known whether hyperlactatemia is correlated with SBE values.[6,7] In this study, we aimed to investigate whether there is a correlation between lactate and SBE values, which are accepted as indirect indicators of tissue perfusion, in patients undergoing cardiac surgery and CPB and to evaluate the intraoperative periods with stronger and weaker correlations.

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Methods

Ethical Approval

This retrospective study was conducted between September 2000 and September 2020. Ethical approval was obtained from the regional ethics committee of our university (ATADEK- 2024-2/72). This study complied with the ethical principles of the Declaration of Helsinki regarding medical research involving human subjects. All patients provided informed consent before the study. This informed consent was obtained verbally.

Patients

In total, 2068 patients >18 years of age who underwent elective cardiac surgery with CPB were included in the study. Patients <18 years of age, with acute–chronic renal failure and acid–base balance disorders, and who underwent cardiac surgery without CPB were excluded.

Digital records of all patients were analyzed (via database analysis), and age, height, weight, body surface area, and bypass and cross-clamp times were recorded. The following hemodynamic and blood gas parameters were recorded at four different time points: before induction of anesthesia (pre-AI), after induction of anesthesia (post-AI), at 36°C at the beginning of ECC, and at the end of ECC (post-ECC):

- Hemodynamic parameters: heart rate, systolic arterial blood pressure, diastolic arterial blood pressure, and mean arterial blood pressure
- Blood gas parameters: pH, partial arterial carbon dioxide and oxygen pressures, arterial oxygen saturation, bicarbonate (HCO_3), SBE, hemoglobin, hematocrit (Hct), sodium, chloride (Cl), potassium, calcium, lactate, glucose, and anion gap.

Anesthesia Management

All patients were admitted to the operating room with 125 µg/kg midazolam IM, 30 min before surgery. Vascular access was established using a 16G IV cannula, and the isotonic solution was started at a rate of 100 mL/h. Hemodynamic monitoring was performed using two-channel electrocardiography (ECG) (DII, V5 leads), pulse oximetry, invasive arterial pressure (right radial artery), and central venous pressure (right internal jugular vein). For anesthesia induction, 50 µg/kg midazolam, 2 mg pancuronium, and 25–35 µg/kg fentanyl were administered. After the administration of pancuronium (0.1 mg/kg), endotracheal intubation was performed.

All patients were started on midazolam and vecuronium infusion at a dose of 80 µg/kg/h and furosemide 0.5 mg/kg as an IV bolus.

After removal of the left internal thoracic artery, the activated clotting time was increased to 450–600 s with the heparin bolus.

CPB was initiated after cannulation. During CPB, Hct, mean arterial pressure, and pump flow were maintained at 25%–30%, 50–80 mmHg, and 2–2.5 L/m^2/min, respectively.

Moderate hypothermia (32°C) was applied to all patients. Following cross-clamping, antegrade cold crystalloid cardioplegia (7–10 mL/kg) was administered. After weaning from CPB, midazolam and vecuronium doses were adjusted to 50 µg/kg/h. After skin closure, midazolam and vecuronium infusions were stopped, and the patient was transferred to the intensive care unit.

Statistical Analysis

Patient characteristics are presented as mean (standard deviation), median (quartiles), and percentage. Pearson's and Spearman's correlation tests were used for correlation analysis, and the kappa test was used to determine SBE and lactate concordance. Statistical analyses were performed using SPSS version 29 software. For statistical significance, $p<0.05$ was accepted.

Results

In total, 2068 patients who underwent cardiac surgery with CPB were retrospectively analyzed. The mean age of the patients was 62±4 years, and 42% were women. The most common surgery was aortocoronary bypass grafting (68%). The mean CPB duration was 67±29

Table 1. Demographic and baseline blood gas and hemodynamic data of patients

Number of patients, n	2068
Age (years)	62±4
Female sex (%)	42
BSA (m^2)	1.36±0.21
Hb (gr/dL)	13.3±1.7
Hct (%)	41.1±5.3
SBE (mmol/L)	0.9±2.1
Lactate (mol/L)	1.0±0.5
PCO_2 (mmHg)	37.8±4.1
PO_2 (mmHg)	82±43
pH	7.42±0.03
Osmolarity (mOsm)	279±8
Anion gap (mmol/L)	8±5
MAP (mmHg)	103±18
HR (bpm)	81±17
CVP (cm/H_2O)	3±4
Surgery type, ACBG (%)	68
Surgery type, valvular surgery (%)	17
Bypass time (min)	67±29
Cross-clamp time (min)	41±20

BSA: Body surface area; Hb: Hemoglobin; Hct: Hematocrit; SBE: Standard base excess; PCO_2: Partial arterial carbon dioxide pressure; PO_2: Partial arterial oxygen pressure; pH: Potential of hydrogen; MAP: Mean arterial pressure; HR: Heart rate; CVP: Central venous pressure; ACBG: Aortocoronary bypass grafting.

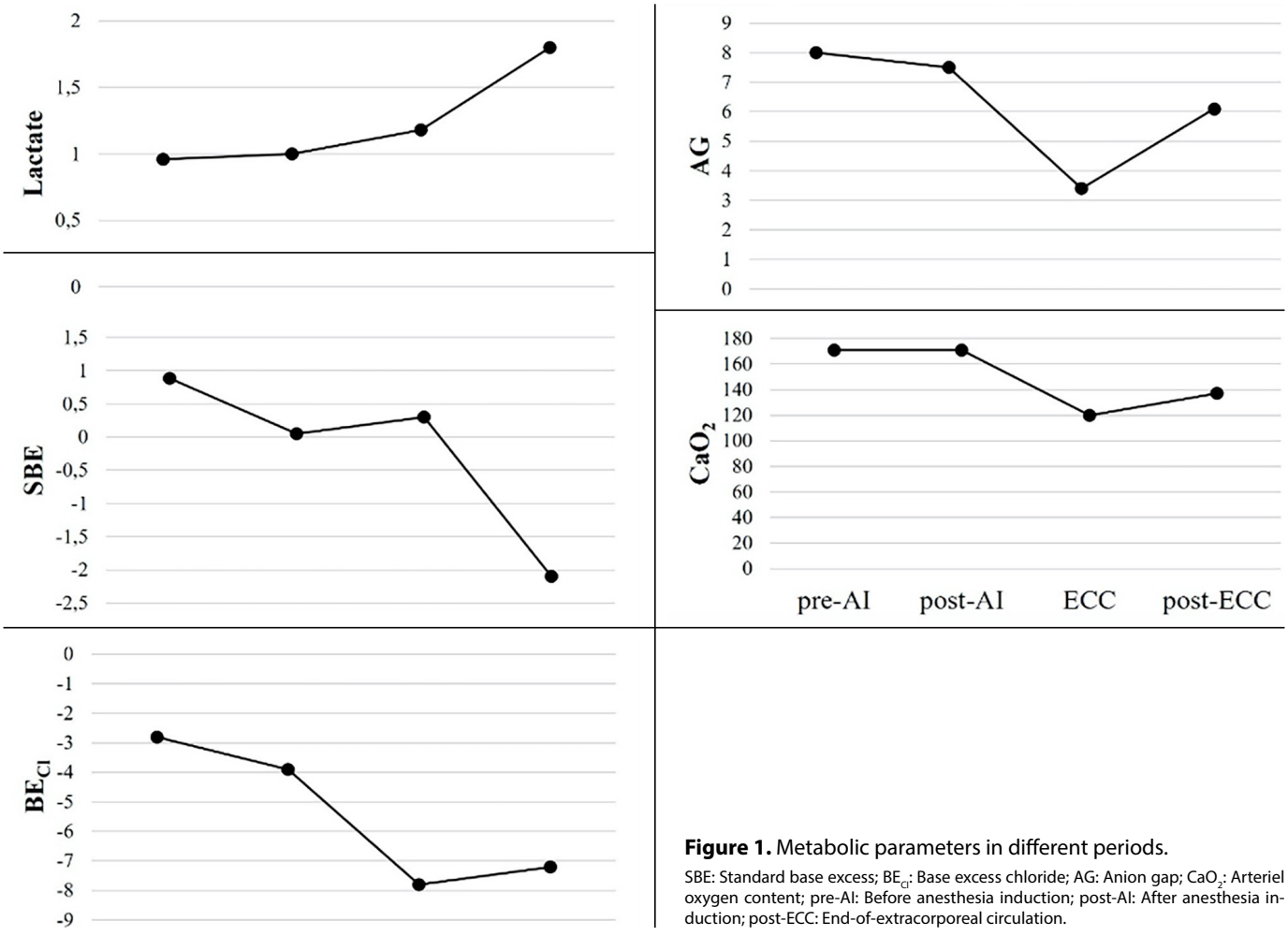

Figure 1. Metabolic parameters in different periods.

SBE: Standard base excess; BE_{Cl}: Base excess chloride; AG: Anion gap; CaO_2: Arteriel oxygen content; pre-AI: Before anesthesia induction; post-AI: After anesthesia induction; post-ECC: End-of-extracorporeal circulation.

min, and the mean cross-clamp time was 41±20 min. The mean lactate level pre-AI was 1.0±0.5 mmol/L, and the mean SBE level was 0.9±2.1. The demographic and baseline blood gas and hemodynamic data of the patients are presented in Table 1.

The intraoperative changes in metabolic parameters (BECl, SBE, lactate, anion gap, and arterial oxygen content) are presented in Figure 1. SBE and lactate values showed a significant but rather weak correlation in all four intraoperative periods (pre-AI, post-AI, ECC, and post-ECC of r^2=0.01, 0.02, 0.06, and 0.06, respectively; p<0.001). The SBE–lactate correlations at different intraoperative periods are presented in Figure 2.

Because of this weak correlation, delta SBE and delta lactate values obtained by subtracting the post-ECC lactate and SBE values from the pre-AI lactate and SBE values to monitor lactate and SBE changes presented a significant but very weak correlation (r^2=0.15; p<0.001; Fig. 3).

When analyzing patients with lactate values >2 mmol/L during the post-ECC period to determine how this correlation changed in patients with hyperlactatemia, we found that SBE and lactate values were correlated (r^2=0.11; p<0.001), but this correlation was not found in patients with a lactate value <2 mmol/L (r^2=0.0003; p=0.526; Figs. 4a, b).

In the lactate–SBE relationship, which was statistically significant but has a weak correlation strength, the kappa test results used to determine whether SBE can be used instead of lactate are presented in Table 2. According to the kappa test results, lactate and SBE changes were incompatible with each other in 274 (13.3%) patients. Hyperchloremia was present in 1683 (94.5%) of 1781 patients with lactate increase and simultaneous SBE decrease. Other blood gas parameters that may affect the lactate–SBE relationship are presented in Table 3.

Discussion

In this study, lactate and SBE, which are considered indirect indicators of tissue perfusion in patients undergoing cardiac surgery with CPB, were weakly correlated during the intraoperative period.

Cardiac surgery using CPB is known to cause acid–base disorders via metabolic changes. Although there are

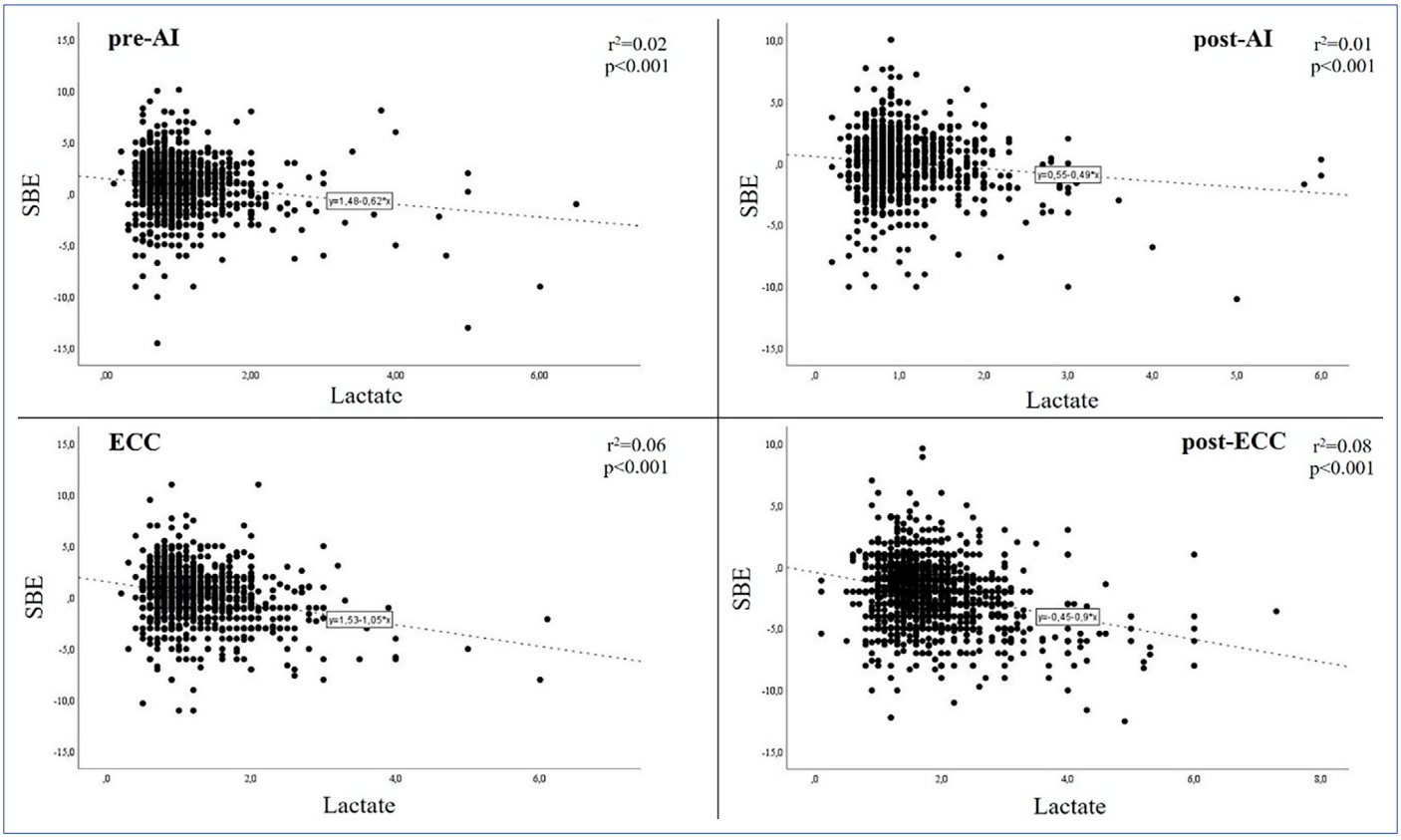

Figure 2. Correlation between SBE and lactate in different periods.

many underlying factors of acid–base disturbances due to cardiac surgery, microcirculatory and macrocirculatory hemodynamic disorders (hemodynamic instability) are the most important, most common, and clinically responsive to treatment.[8] Therefore, blood lactate and SBE are the most commonly used parameters for detecting tissue perfusion disorders caused by hemodynamic instability in clinical practice.

Elevated blood lactate levels associated with metabolic acidosis are common in patients with systemic hypoperfusion and tissue hypoxia.[9] This condition represents type A lactic acidosis resulting from an imbalance between tissue oxygen supply and demand.[10,11] Type A hyperlactatemia is common among patients undergoing cardiac surgery, and type B hyperlactatemia caused by factors other than tissue hypoxia may also occur.[12] Many studies have indicated that in the presence of tissue perfusion impairment in patients undergoing cardiac and noncardiac surgery, blood lactate levels increase, which has prognostic value.[8,13,14]

Maillet et al.[15] showed that nonelective surgery, prolonged CPB, and intraoperative vasopressor use were independent risk factors for early hyperlactatemia after cardiac surgery and further showed that early hyperlactatemia predicts intensive care unit mortality more accurately than late hyperlactatemia. Weil et al.[16]

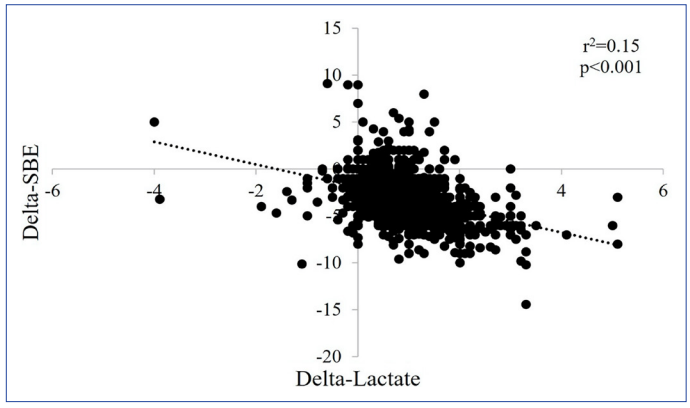

Figure 3. Correlation between delta-SBE and delta-lactate.

showed that when lactate levels increased from 2.0 to 8.0 mmol/L, the probability of survival decreased from 90% to 10%, but this prognostic inference depended on the cause of the increase in lactate levels.

Hyperlactatemia can occur with or without concomitant metabolic acidosis. When hyperlactatemia occurs in the presence of good tissue perfusion, such as increased metabolic activity due to catecholamine administration, alkalosis, sepsis, or burns, buffering mechanisms can compensate for any decrease in pH. However, when lactate levels increase because of poor tissue perfusion, buffering systems cannot manage the increase and acidosis may develop.

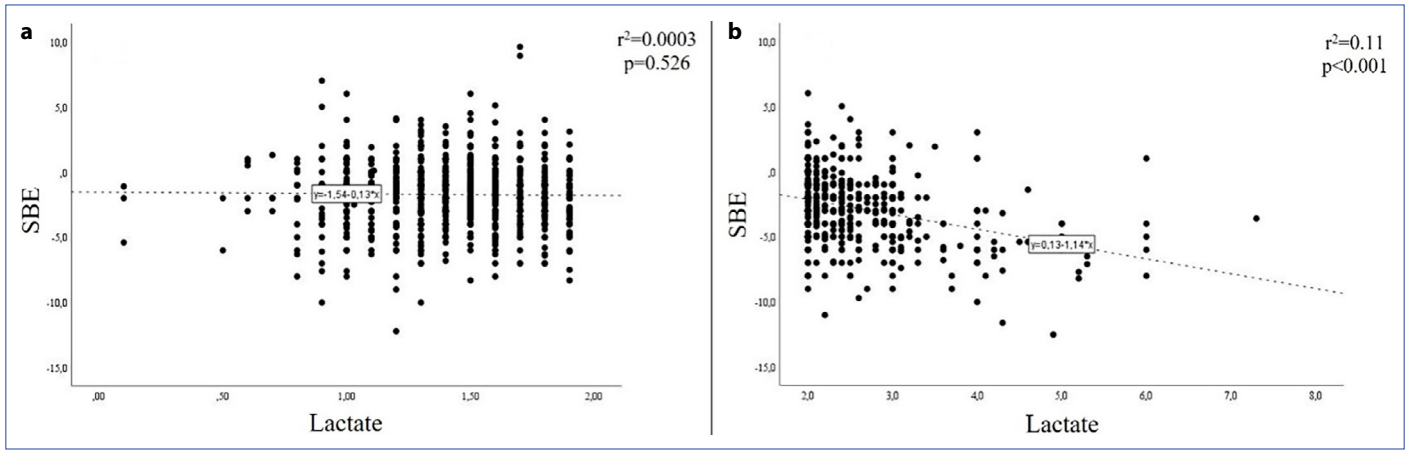

Figure 4. (a,b) Correlation between SBE and lactate in patients with post-ECC lactate levels of **(a)** <2 mmol and **(b)** ≥2 mmol.

Table 2. Compatibility between SBE and lactate changes

Kappa: 0.07 p<0.001
Spearman correlation: 0.004 p=0.859

	No lactate change		Lactate increase		Lactate decrease		Total	
	n	%	n	%	n	%	n	%
No SBE change	8	6.7	45	2.4	6	10.5	59	2.9
SBE decrease	105	87.5	1781	94.2	46	80.7	1932	93.4
SBE increase	7	5.8	65	3.4	5	8.8	77	3.7
Total	120	100.0	1891	100.0	57	100.0	2068	100.0

Changes were calculated by subtracting the Post-ECC values from the Pre-AI values. Post-ECC: End-of-extracorporeal circulation; Pre-AI: Before anesthesia induction.

Table 3. Comparison of intraoperative blood gas and hemodynamic parameters in different periods

	Pre-AI	Post-AI	ECC	Post-ECC	p
pH	7.42 (7.41–7.44)	7.42 (7.40–7.45)	7.44 (7.41–7.47)^{ßßß}	7.39 (7.36–7.42)$^{###}$	<0.001
PaO_2, mmHg	76 (68–85)	206 (171–240)***	273 (178–385)^{ßßß}	201 (138–270)$^{###}$	<0.001
$PaCO_2$, mmHg	38.0 (35.6–40.0)	36.0 (34.0–39.0)***	35.0 (32.3–38.0)^{ßßß}	36.0 (33.0–39.0)$^{###}$	<0.001
HCO_3, mmol/L	25.0 (24.0–26.0)	24.4 (23.6–25.8)***	25.0 (24.0–26.0)^{ßßß}	22.7 (21.0–24.0)$^{###}$	<0.001
SBE, mmol/L	1.0 (−0.2; 2.0)	0.0 (−1.0; 1.0)***	0.5 (−1.0; 1.6)^{ßßß}	−2.0 (−3.1; −1.0)$^{###}$	<0.001
Hb, gr/dL	13.0 (12.0–14.1)	13.0 (12.0–14.0)	9.0 (8.0–10.0)^{ßßß}	10.0 (9.0–11.6)$^{###}$	<0.001
Hct, %	41.0 (38.0–45.0)	40.0 (36.0–43.6)	28.0 (25.0–31.0)^{ßßß}	32.0 (29.0–36.0)$^{###}$	<0.001
SaO_2, %	96 (95–97)	100 (100–100)***	100 (99–100)	100 (99–100)	<0.001
Lactate, mol/L	0.9 (0.7–1.0)	0.9 (0.7–1.1)	1.0 (0.9–1.3)	1.7 (1.3–2.0)$^{###}$	<0.001
Na, mmol/L	137 (135–139)	137 (135–139)	134 (132–135)^{ßßß}	136 (134–137)$^{###}$	<0.001
K, mmol/L	3.9 (3.7–4.1)	3.9 (3.7–4.1)	3.9 (3.6–4.1)	3.9 (3.6–4.1)	0.135
Cl, mmol/L	107 (105–110)	108 (106–111)	110 (108–112)^{ßßß}	111 (108–113)	<0.001
Ca, mmol/L	1.1 (1.1–1.2)	1.1 (1.1–1.1)	1.1 (1.1–1.2)	1.2 (1.1–1.3)$^{###}$	<0.001
Glucose, mgr/dL	105 (96–127)	110 (99–129)***	108 (94–130)	147 (126–179)$^{###}$	<0.001
HR, bpm	80 (70–90)	82 (70–95)	–	90 (80–100)$^{$$$}$	<0.001
MAP, mmHg	101 (91–113)	84 (75–93)***	65 (53–77)^{ßßß}	81 (74–89)$^{###}$	<0.001

The Friedman test was used for all group comparisons, and the Wilcoxon test was used for pairwise comparisons. ***: Pre-AI and post-AI period comparisons; ^{ßßß}: Post-AI and ECC period comparisons; $^{###}$: , ECC and post-ECC period comparisons; $^{$$$}$: Post-AI and ECC period comparisons for HR. pre-AI: Before anesthesia induction; post-AI: After anesthesia induction; post-ECC: End-of-extracorporeal circulation; HCO_3: Bicarbonate; SaO_2: Arterial oxygen saturation; Na: Sodium; K: Potassium; Cl: Chloride; Ca: Calcium.

Although increased lactate levels and associated factors during cardiac surgery have been extensively studied, the prognostic role of SBE changes and whether SBE changes are correlated with lactate levels are unclear.[17,18] Whether base excess (BE) or lactate levels have better prognostic abilities for mortality and morbidity remains controversial. Two studies in trauma patients showed that BE was a superior predictor of mortality than lactate, and another study in a mixed surgical intensive care unit cohort showed no difference between lactate and BE in their ability to predict mortality.[19–21] Zante et al.[7] showed that BE below –6.7 mmol/L on intensive care unit admission was the only predictor of ICU mortality in a large cohort of patients undergoing cardiac surgery.

In this study, lactate levels were correlated with SBE throughout the intraoperative period, but the strength of the correlation was very weak. To understand and interpret the correlation between lactate and SBE values, it is important to know how SBE is calculated and the variables that contribute to the calculation.

SBE was formulated by Siggaard Andersen as $SBE=0.9287\times(HCO_3-24.4+14.83\times(pH-7.4))$ by revising Van Slyke's equation.[22] In the early 2000s, O'Dell and Story proposed a partitioned BE model, which claimed that SBE is equal to the sum of the effects of chlorine, lactate, albumin, and unmeasured anions on BE, and they named these parameters BE chlorine, BE lactate, BE albumin, and BE unmeasured anion, respectively: SBE=BECl+BEAlb+BElactate+BEUA.[23,24] According to these equations, SBE is a calculated and dependent parameter. In 2021, chlorine, lactate, albumin, and unmeasured anions were shown to be independent variables for SBE.[25] Therefore, all of these parameters can independently change the SBE. Therefore, SBE is expected to change with lactate changes; however, changes in other parameters also affect SBE. The weak SBE–lactate correlation and low kappa coefficient in this study can be attributed to the effects of other parameters such as chlorine, lactate, albumin, unmeasured anions on SBE. Predicting lactate changes using SBE has two drawbacks.

1. Although lactate changes occur, SBE may not exhibit them. According to the kappa test, lactate and SBE changes were discordant in 274 (13.3%) patients.
2. Even if lactate and SBE changes are compatible, it cannot be said with certainty that only lactate change is the cause. According to the kappa test, 1683 (94.5%) of 1781 patients with lactate increase and concomitant SBE decrease also had hyperchloremia, and both were responsible for the SBE decrease.

Therefore, the use of SBE alone to predict possible metabolic changes is inadequate. The correct approach is to measure lactate levels and interpret their effects on SBE. Weak correlations and low kappa coefficients indicate that SBE alone cannot predict lactate changes.

Conclusion

These results suggest that during cardiac surgery with CPB, blood lactate level monitoring is still the most valuable parameter for identifying patients with the potential to deteriorate and associated adverse outcomes, and SBE monitoring alone may be insufficient.

Disclosures

Ethics Committee Approval: The study was approved by The Acıbadem Mehmet Ali Aydınlar University Medical Research Ethics Committee (no: 2024-2/72, date: 15/02/2024).

Authorship Contributions: Concept – F.T., BG.; Design – F.T., S.A.Y.; Supervision – F.T., B.G.; Data collection &/or processing – S.A.Y., M.T.C.; Analysis and/or interpretation – B.G., M.T.C.; Literature search – S.A.Y., M.T.C.; Writing – S.A.Y.; Critical review – F.T., B.G.

Informed Consent: Written informed consent was obtained from all patients.

Conflict of Interest: All authors declared no conflict of interest.

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